reported in California as of January 1, 1989, nearly 90% were gay or bisexual men and 4% were intravenous drug abusers.

The overall prevalence of HIV infection in western states continues to be low. For example, 0.016% of 1,550,693 units of whole blood donated in California between July 1987 and September 1988 was HIV seropositive. For the quarter ending September 1988, the HIV-seropositivity rate was 0.013%, which was down from a rate of 0.13% in the summer of 1985. Likewise, results from testing all babies born in California in July 1988 showed that only 36 of 43,301 (0.083%) had antibodies indicative of maternal HIV infection.

Data from acute care health facilities serving disproportionate numbers of higher risk persons indicate a relatively higher rate of infection. A 1987 HIV-seroprevalence survey of various types of publicly funded health facilities in 21 California counties—excluding San Francisco and Los Angeles counties—found 2.1% of 5,282 persons tested to be HIV seropositive, with the rate ranging from 1.1% in primary care public health clinics to 3.1% in sexually transmitted disease clinics. Similarly, the results of confidential HIV testing at 16 publicly funded family planning and primary care clinics from January through June 1988 showed 1.9% of 6,390 persons seropositive—0.33% of females and 6.04% of males. Further, 2.5% of 5,372 men and 3.1% of 807 women entering California's state prison system in the spring of 1988 were HIV positive. In each of these studies almost all of the seropositive persons belonged to recognized high-risk groups.

Despite the generally low prevalence of HIV infection in western states, the possibility of encountering HIV-infected persons in the course of emergency medicine practice and the consequences of acquiring the infection require that emergency medical personnel know the epidemiology and clinical manifestations of HIV disease and understand the indications for and complexities of HIV antibody testing. Emergency medical personnel also need to rigorously adhere to established infection control procedures, which assume that all patients are potentially infectious for HIV or other bloodborne pathogens.

Emergency medical personnel must assiduously guard against accidental needle sticks because they present the greatest risk for health care work-related HIV infection. To date, about 80% of all health care workers suffering work-related HIV infection have acquired the infection from needle sticks. National data indicate that the risk of acquiring HIV infection following a needle stick contaminated with blood from a known HIV-infected person is approximately 0.5%. Health care personnel have also contracted HIV infection as a result of infected blood splashing on or otherwise contacting mucous membranes or open lesions.

All emergency services should have comprehensive ongoing programs to reduce accidental needle sticks and blood exposures; training in universal body substance precautions; specified policies and procedures for voluntary and confidential HIV antibody testing of patients from whom possible exposure to HIV may have occurred, as well as for exposed workers; and a comprehensive postexposure program, including counseling and consideration of the prophylactic use of zidovudine (AZT).

All health care personnel with exposure to blood from needle sticks should be reported as soon as possible to the Hospital Infections Program at the Centers for Disease Control (CDC), telephone number 1 (404) 639-1644, for possible enrollment in a postexposure AZT study and to develop a better data base for assessing risk. A baseline blood specimen also should be drawn as soon as possible after exposure and sent to the CDC. Similarly, Burroughs Wellcome Company is enrolling participants in a placebo-controlled study of the efficacy of the prophylactic use of AZT for health care workers who have sustained a needle stick (telephone, 1-800-HIV-STIK).

Consistently adhering to universal infection control procedures and addressing other HIV-related issues and concerns may be challenging in the emergency care setting and, therefore, must be anticipated and planned for.

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Paralyzing Agents in the Emergency Department

NEUROMUSCULAR BLOCKING DRUGS have been used for many years in operating rooms to assist intubation and provide muscular relaxation. In the past five years, paralyzing agents have achieved increasing use in emergency departments and have been introduced into the prehospital care setting. Paralyzing drugs are valuable in assisting endotracheal intubation in patients with head injuries, multiple trauma, drug overdose, and status epilepticus. They are also of benefit to patients who must be rendered motionless while undergoing emergent diagnostic tests such as computed tomographic (CT) scans or invasive procedures. Recent studies support the continued use of paralyzing agents in emergency departments.

Paralyzing drugs fall into two classes. The depolarizing neuromuscular blockers cause an initial depolarization at the neuromuscular end plate, characterized by muscular fasciculations. This is followed by complete irreversible muscular paralysis within 60 seconds, which lasts 2 to 5 minutes. The most commonly used depolarizing blocker is succinylcholine chloride. The dose in children and adults is 1 to 1.5 mg per kg body weight by intravenous push.

There are a number of serious adverse effects associated with using succinylcholine, including severe bradycardia, ventricular arrhythmias, and hyperkalemia. Other complications stem from fasciculations, such as regurgitation and aspiration, displacement of fractures, new fractures in osteopenic patients, and increases in intraocular pressure. Most possible complications of succinylcholine use can be avoided by carefully screening patients and using premedication. Administering atropine sulfate will prevent bradycardia; a priming dose of a nondepolarizing neuromuscular blocker will prevent fasciculations.

The nondepolarizing muscle relaxants (NDMRs) bind to the acetylcholine receptor at the neuromuscular end plate and competitively inhibit acetylcholine. They first cause muscular weakness followed by total paralysis within two to three

minutes. Fasciculations do not occur. Paralysis lasts from 40 minutes to about 3 hours, depending on the dose and agent used. Paralysis may be reversible with the use of cholinesterase inhibitors such as neostigmine. The effects of NDMRs may be notably prolonged in patients with neuromuscular diseases such as myasthenia gravis. The NDMR drugs most commonly used are pancuronium bromide, atracurium besylate, and vecuronium bromide. Administering pancuronium (dose, 0.1 mg per kg) produces paralysis lasting about 60 minutes. The effects of atracurium (0.5 mg per kg given intravenously) and vecuronium (0.1 to 0.15 mg per kg intravenously) last 25 to 40 minutes. Vecuronium may be the ideal NDMR for emergency department use because it is short acting and causes minimal histamine release and hemodynamic changes.

Since 1986 at least four studies have been published to document the safe and effective use of neuromuscular blocking drugs in emergency departments. Emergency care physicians typically use succinylcholine to assist intubation in the subset of patients noted. The NDMR drugs are used to maintain paralysis in intubated patients and to induce paralysis in patients who have head CT scans or invasive procedures. Despite the fact that paralyzing drugs were used almost exclusively in critically ill patients in these studies, reported serious complications were rare.

There are a number of prerequisites to the efficient, safe use of neuromuscular blockers by emergency staff. First, physicians must be expert in airway management, including the rare instance when a cricothyrotomy will be necessary. Patients who might be difficult to intubate and ventilate after paralysis—that is, those with epiglottitis, massive facial trauma, or airway obstruction—are not candidates for the use of paralyzing drugs. Each of the centers reporting success and safety with paralyzing drugs mandated special training by physicians and nurses in the pharmacology and complications of neuromuscular blocking drugs. Attention was given to premedication to minimize succinylcholine side effects, careful cardiac monitoring of patients during intubation, sedation of awake patients who were paralyzed, and assuring adequate oxygenation and ventilation once paralysis occurred.

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Pulse Oximetry in Emergency Medicine

PULSE OXIMETRY is a noninvasive and continuous measurement of oxygen saturation (Sao_2). Oxygen saturation is measured by placing a special sensor on a patient's finger, toe, or nose. This sensor consists of a light-emitting diode—which projects two discrete wavelengths of light corresponding to saturated and unsaturated oxyhemoglobin—and a photodetector.

During the diastolic phase of a patient's pulse wave, a reference intensity of light is discerned. During systole the light intensity will vary with the amount of oxygenated hemoglobin present in the blood. Because the measurements are made using the change in light absorption between systole and diastole, no interference is present from anatomic aspects other than hemoglobin. Unlike its predecessor, the transcutaneous oxygen monitor, the pulse oximeter does not need to be "warmed up" or calibrated.

Oximetry was first conceptualized in the 1930s, but Leland Clark's introduction of the polarographic oxygen electrode in 1954 paved the way for the present-day use of arterial blood gas pressures and the measurement of the partial pressure of oxygen (Pao_2). In 1983 oximetry was reintroduced in the form of pulse oximetry, and the technique rapidly gained initial acceptance through the monitoring of patients during anesthesia at risk for hypoxia. Since then clinical applications have expanded dramatically and presently include all patients at risk for hypoxia—such as those with respiratory failure, asthma, and endotracheal tubes in place—who would benefit from continuous oxygen saturation monitoring. The adequacy of airway management is also aided by pulse oximetry.

Many clinical and experimental studies support the fact that the noninvasive determination of Sao_2 has an accuracy of $\pm 2\%$; values of Sao_2 in the range of 70% to 99% are comparable to invasively derived laboratory values. Oxygen saturation, however, is related to the Pao_2 by the oxyhemoglobin dissociation curve, and patients who have an Sao_2 of 90% will have a resultant Pao_2 of 60 mm of mercury. Therefore, a small drop in the Sao_2 in the range of 90% to 99% will reflect a large drop in the corresponding Pao_2. In addition, it should be remembered that pulse oximetry will be inaccurate in patients with hypothermia ($<35°C$), hypotension (<50 mm of mercury), severe vascular disease, vasopressor therapy, abnormal pigmentation of the blood (such as bilirubin), or with abnormal hemoglobin (such as carboxyhemoglobin and methemoglobin).

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Thrombolytic Therapy for Acute Pulmonary Embolism

EVEN WITH prompt treatment, acute pulmonary embolism is associated with substantial morbidity and mortality. There has been considerable confusion regarding the indications for using heparin or thrombolytic agents for treating pulmonary embolism. Recent literature has, however, helped to clarify the role for each of these therapies and has emphasized the increased use of thrombolytic agents.

Thrombolytic therapy for acute pulmonary embolism has been shown to lyse clots, normalize pulmonary artery pressures, and restore pulmonary vascular perfusion faster than heparin therapy. Despite this clinical evidence, thrombolytic therapy remains underused. Although early studies suggested no difference in mortality between patient groups receiving thrombolytic therapy or heparin, more recent literature suggests there is a particular subset of patients who are most likely to benefit from thrombolytic therapy. This includes patients with massive pulmonary embolism involving two or more lobar arteries, pulmonary embolism with hemo-